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Renal medullary solute depletion resulting from psychogenic polydipsia in a rhesus monkey

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A 9-year-old 9-kg male rhesus monkey was observed to have polydipsia and an unthrifty appearance. The monkey was being fed a commercial primate ration, with fresh water provided ad libitum by automatic waterer, and was housed in individual caging. Physical examination revealed extensive bilateral periorbital edema and patchy alopecia over the lower trunk and limbs. Blood cell counts were unremarkable, but serum biochemical analysis revealed hyponatremia ($116 \text{ mEqNa}^+/\text{L}$; normal, 145 to 158 mEq/L)¹ and hypochloremia ($81 \text{ mEqCl}^-/\text{L}$; normal, 106 to 115 mEq/L)¹ and the plasma osmolality was calculated to be 247 mosm/kg of H_2O (normal, 295 to 330 mosm/kg of H_2O).¹⁻² Urinalysis revealed a specific gravity of 1.000 .

The only environmental alteration recorded for this monkey was a change in feeding regimen. The monkey had been inadvertently fed almost twice his daily requirement of biscuits for nearly 4 months, resulting in obesity. Four weeks before the onset of polydipsia, the diet was restricted to 100% of the calculated daily caloric requirement, to diminish further unnecessary weight gain. This apparently was the triggering factor for the polydipsia.

The monkey's water consumption was monitored while he was given free access to bottled water. The monkey's mean daily water consumption was 3.75 L , or 417 ml/kg of body weight, which is approximately 5 times the normal water consumption for a rhesus monkey fed a commercial dry diet.³ To establish the cause of the hyposthenuria, a water deprivation test was performed. The urine specific gravity after 10 hours was 1.000 . This lack of renal concentrating ability indicated either a lack of anti-diuretic hormone (ADH) production or diminished renal responsiveness to ADH. Although renal failure was considered, normal BUN and creatinine values, lack of anemia, and normal hydration tended to rule it out. An ADH stimulation test was performed, with 5 U of aqueous vasopressin administered intramuscularly. Urine specific gravity was monitored hourly for 5 hours and again did not exceed 1.000 . The lack of ability to concentrate urine after exogenous adminis-

tration of ADH would normally indicate nephrogenic diabetes insipidus; however, one would expect the serum osmolality to be greater than normal.⁴

On the basis of the low serum osmolality, renal medullary solute depletion was suspected. To verify the diagnosis, water intake was restricted to 1.5 L/day , and the monkey was closely monitored. Signs of distress or dehydration were not observed. The urine specific gravity remained at 1.000 . After 7 days of restricted water intake, the ADH stimulation test was repeated and resulted in a urine specific gravity of 1.010 . Also, the serum electrolyte values had returned to within normal limits. The water deprivation test was repeated 3 days later and resulted in a urine specific gravity of 1.020 . Water intake was restricted to 1 L daily for 2 months, during which time serum electrolyte concentrations remained normal during weekly monitoring. Initially, urine specific gravity remained constant at 1.000 . On day 5, the specific gravity was 1.005 , and continued to slowly increase until values ranged from 1.015 to 1.025 on a daily basis. At the end of the 2 months, a water deprivation test resulted in an increase in urine specific gravity from 1.020 to 1.030 within 2 hours.

The normal response to water deprivation verified a normal concentrating ability and the diagnosis of psychogenic polydipsia with renal medullary solute depletion. One year after diagnosis and treatment, there was no evidence of recurrent polydipsia.

Various abnormal behaviors, including foot-thumb-sucking, cage-chewing, abnormal vocalization, aggression, and polydipsia have been attributed to cage confinement of nonhuman primates.⁵⁻⁹ Renal medullary solute depletion develops when prolonged diuresis results in a loss of the normal solutes, including urea, from the medullary interstitium.¹⁰ When the osmotic gradient between the medulla and the cortex decreases, the renal concentrating ability decreases concurrently, resulting in the inability to respond to either water deprivation or ADH stimulation. The rhesus monkey kidney does not have a well developed inner medulla with long loops of Henle.¹¹ It has been suggested that because of this anatomic variation, urine formation in rhesus

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monkeys is not so dependent on the medullary osmotic gradient as in other species.¹¹ However, in this case, medullary solute depletion did develop in response to prolonged diuresis, and resulted in a lack of renal concentrating ability.

Reversal of renal medullary solute depletion may be attempted by gradual water restriction with concurrent monitoring of hydration, urine specific gravity, and BUN concentration.^{12,13,14} In this case, after 1 week of water restriction to 1500 ml/day, there was a minimal response to ADH administration because of restoration of hypertonicity to the renal medullary interstitium. Correction of renal medullary solute depletion must be accomplished before the cause of polyuria may be established.¹⁵

1. McClure HM. Hematologic, blood chemistry, and cerebrospinal fluid data for the rhesus monkey. In Bourne GH, ed. *The rhesus monkey. Vol II. Management, reproduction, and pathology*. New York: Academic Press Inc, 1975:409-429.
2. Scott RC. Disorders of sodium metabolism. *Vet Clin North Am (Small Anim Pract)* 1982;12:375-397.
3. Richter CB, Lehner NDM, Henrickson RV. Primates. In Fox JG, Cohen BJ, Loew FM, eds. *Laboratory animal medicine*. Orlando, Fla: Academic Press Inc, 1984:297-383.

4. Grauer GF, Grauer RM. Veterinary clinical osmometry. *Compend Contin Educ Pract Vet* 1983;5:539-544.
5. Hardy RM. Disorders of water metabolism. *Vet Clin North Am (Small Anim Pract)* 1982;12:353-373.
6. Mulnix JA. Diabetes insipidus. In Kirk RW, ed. *Current veterinary therapy VIII, small animal practice*. Philadelphia: WB Saunders, 1983:850-851.
7. Lage AL. Apparent psychogenic polydipsia. In Kirk RW, ed. *Current veterinary therapy VI, small animal practice*. Philadelphia: WB Saunders, 1977:1098-1102.
8. Cross HA, Harlow HF. Prolonged and progressive effects of partial isolation on the behavior of macaque monkeys. *J Exp Res Pers* 1965;1:39-49.
9. Spineili JS, Markowitz H. Prevention of cage-associated distress. *Lab Anim* 1985;14:19-28.
10. Grauer GF. The differential diagnosis of polyuric-polydipsic diseases. *Compend Contin Educ Pract Vet* 1981;3:1079-1086.
11. Tisher CC. Structure and function of the rhesus kidney. In Bourne GH, ed. *The rhesus monkey. Vol I. Anatomy and physiology*. New York: Academic Press Inc, 1975:107-143.
12. Cotter SM. Polyuria and polydipsia. In Ettinger SJ, ed. *Textbook of veterinary internal medicine*. 2nd ed. Philadelphia: WB Saunders, 1983:133-138.
13. Brettschwerdt EB. Clinical abnormalities of urine concentration and dilution. *Compend Contin Educ Pract Vet* 1981;3:414-422.
14. Low DG. Polyuric renal failure. In *Proceedings 3rd Annu Med Forum* 1985;3:17-20.

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